“CLINICAL DETERMINANTS OF CORONARY COLLATERALS AN ANGIOGRAPHY STUDY”

Dissertation submitted to
The Tamil Nadu Dr. M.G.R Medical University, Chennai
in partial fulfillment of the requirements for the degree of

DM Cardiology
Branch II
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MADRAS MEDICAL COLLEGE

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CERTIFICATE

This is to certify that **Dr. PREMANANTH .N**, Post graduate student [2010-2013] in the Department of Cardiology, Government General Hospital, Chennai & Madras Medical College, Chennai-600003, has done this Dissertation on **"CLINICAL DETERMINANTS OF CORONARY COLLATERALS AN ANGIOGRAPHY STUDY"** under my guidance and supervision in partial fulfilment of the regulations laid down by The Tamil Nadu Dr. M.G.R Medical University, Chennai, for DM Cardiology –Branch II examination to be held in August, 2013.

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DECLARATION

I hereby solemnly declare that the dissertation titled **"CLINICAL DETERMINANTS OF CORONARY COLLATERALS AN ANGIOGRAPHY STUDY"** was done by me at Department of Cardiology, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai-3 during 2012 under the guidance and supervision of my unit Chief Prof. Dr.V.E.DHANDAPANI, MD,DM.

The dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical University towards the partial fulfillment of requirement for the award of D.M. (Branch-2) in Cardiology.

Place: **SIGNATURE OF THE CANDIDATE**

Date :

(Dr. PREMANANTH .N)

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“CLINICAL DETERMINANTS OF CORONARY COLLATERALS”

AN ANGIOGRAPHIC STUDY

CONTENTS

ABBREVIATIONS

ACS- Acute coronary syndrome

CSA- chronic stable angina

CAD- Coronary artery disease

DM- Diabetes mellitus

ECG- electrocardiogram

ECHO- echocardiogram

LAD- left anterior descending artery

LCX- left circumflex artery

OM –obtuse marginal branch

PDA- posterior descending artery

PLB- posterolateral branch

RCA- right coronary artery

SHT- Systemic hypertension

INTRODUCTION

Coronary artery disease (CAD) is the leading cause of death in developed countries and may become the most important reason for mortality in the developing countries. Early interventions of these patients with CAD by improving anginal symptoms, coronary blood flow with anti-anginal medication, mechanical intervention as percutaneous angioplasty and coronary stenting or coronary artery bypass grafting can minimize the mortality and morbidity. The aim of the intervention is to reduce cardiovascular mortality by reducing the myocardial infarction size and to improve the left ventricular function. However, in extensive CAD, 20–30% of patients may not be suitable for revascularization as angioplasty or surgical bypass and carry a poor prognosis. Hence an alternative treatment strategy for revascularization that improves symptoms as well as the progression of CAD becomes essential.

In normal persons without significant CAD, myocardial blood flow is by the major coronary arterial branches. Side branches originate from the main branch and terminate as an extensive capillary network supply different regions of the myocardium. When one of the major pathways is severely obstructed by an atherosclerotic lesion, flow distal to the lesion is compromised. Functional and structural capacity of myocardium

depends on the adequacy of flow through the alternate coronary pathways or coronary collaterals.

Coronary collaterals are anastomotic connections without an intervening capillary bed between branches of the same coronary artery and between different coronary arteries [(1)]. Collateral circulation has been reported in literature to protect and preserve myocardium around the time of coronary occlusion and improve residual myocardial contractility and left ventricular function. Fukai et al found that well-developed coronary collaterals may minimize the infarct size and increase the amount of viablemyocardium.[2] Sabia et al demonstrated that the myocardium may remain viable in the presence of a good collateral circulation for a prolonged period in patients with acute myocardial infarction with an occluded infarct-related coronary artery [3]. Fulton and Royen demonstrated that in coronary artery obstruction, functional coronary collaterals develop by enlargement of pre-existing coronary anastomotic channels between side branches of two coronary arteries.[4] Most observers agree that more than 90% of occlusion of coronary artery is necessary to bring changes in the new vessel formation. Current studies have shown that vasculogenesis formation in fetus does not play a significant role in adult collateralization. Angiogenesis and arteriogenesis are the known mechanisms of coronary collateral development.

There is lot of studies regarding the presence of collaterals, the pathogenesis of collaterals, individual risk factors, But there is a paucity in literature about the combined risk factors, correlation of clinical presentation to the formation of collaterals. further there is scarcity of studies for grading of collaterals in all the risk factors and clinical presentations. Hence the knowledge of various variable and its association with collateral formation and grade of collateral may provide an insight in modifying the treatment modality.

This study is carried out to find the clinical determinants of coronary collateral in patients with total or subtotal occlusion on coronary angiography and to correlate with grading of collaterals. The information obtained may add and cryatallize to the testimony of others to make it a fruitful wisdom.

AIMS AND OBJECTIVES

1. To study the impact of type of coronary artery disease in coronary collateral development.

2. To study the role of conventional coronary risk factors in the coronary collateral development.

3. To study the coronary collateral number, types and its relationship with the coronary artery involved in total and subtotal coronary artery occlusion.

REVIEW OF LITERATURE

Coronary artery disease(CAD) is a commonly occurring disease accounting for nearly 30% of all deaths worldwide[6] .CAD has been classified as acute coronary syndrome, sudden cardiac death, chronic CAD. Acute coronary syndrome represents conditions which have a common end result, acute coronary ischemia which is usually caused by atherosclerotic plaque rupture, fissuring, erosion with or without superimposed thrombus. This includes acute MI as STEMI [ST elevation MI] or NSTEMI [non ST elevation MI] and unstable angina.

Conventional Risk Factors

There has been many conventional atherothrombosis risk factors known which are associated with CAD, which includes smoking, systemic hypertension, hyperlipidemia, insulin resistance and diabetes, physical activity, and obesity. Novel atherothrombotic risk markers, including high-sensitivity C-reactive protein (hsCRP) and other markers of inflammation, as well as homocysteine and lipoprotein(a). [6]

Coronary circulation

Coronary arteries are the major arterial supply to heart. Aortic sinus gives off two major coronary arteries which subdivide on the

epicardial surface of heart. Left main coronary artery arises from left aortic sinuses and right coronary artery arises from right aortic sinuses. left main coronary artery after its origin, divides into anterior descending and circumflex artery.[7]

Both coronary arteries gives off small, deeply penetrating branches and an extensive network of intramural vasculature as arteries, arterioles and capillaries (5). Small intramural collateral vessel connects the coronary arteries which get enlarged after coronary obstruction. Venous drainage of the Heart is via coronary sinus and thebesian veins.

The resting normal coronary blood flow is between 60 to 90 ml/min/100gm of myocardium and with a potential to augment flow 5 times rapidly during exercise. The coronary flow rate is mainly determined by coronary perfusion pressure and vascular resistance within and outside the coronary vascular bed. The control of coronary blood flow can be metabolic [auto regulation],mechanical ,autonomic and endothelial control .[7]

Coronary Collaterals:

Inspite of so much understanding about coronary circulation in normal hearts, the knowledge about development of coronary collateral

circulation is still not clear. In any arterial occlusion, affected organ may be less sensitive to episodes of ischemia if it is supplied with well established collateral vessels. The potential of an individual to develop coronary collateral is often neglected. Fukai et al found that a well established coronary collaterals minimizes the ischemic area supplied by occluded vessels[2]. In coronary artery occlusion, when original vessel fails to provide sufficient blood supply, Coronary collaterals circulation forms an important alternative source of blood supply. Coronary collaterals are the communicating channels between the major coronary arteries and their branches ().This anastomotic connections do not have an intervening capillary bed between portions of the same coronary artery and between different coronary arteries [1]

History about literature on coronary collateral dates back to more than 200 years ago, when Heberden astonishingly described a patient who got nearly cured of his angina pectoris by sawing wood each day . [8] He described this phenomenon as warm up" or "first effort angina" and ascribed this to coronary vasodilation mainly due to opening of coronary collateral vessels to support the ischemic myocardium. More recent literature reviews interpret this type of "walk through angina" as a biochemical Ischemic preconditioning rather than due to collateral

recruitment . Both mechanisms may probably contribute to the described phenomenon.

In 1956, Baroldi et al demonstrated the presence corkscrew-shaped collaterals at birth in normal human hearts (while few other authors demonstrated coronary collaterals in animals.[9] Baroldi demonstrated collateral diameter of 20 to 350 microns and lengths ranging from 1 to 5 cm in human, whereas schaper and others, demonstrated coronary collaterals lumen greater than 50 microns in dog and less than 50 microns in pig. In dog the collaterals were found predominantly in epicardial layer, whereas in pig the collaterals were demonstrated in intramyocardial and subendocardial layers [10].

Development of coronary collaterals

Angiogenesis and Arteriogenesis are suggested as the mechanisms of coronary collateral development.

Angiogenesis is a term formerly used to describe the formation of new capillaries by sprouting out from preexisting postcapillary venules.

Arteriogenesis is the growth and development of the collateral circulation in the form of the transformation of preexisting arterioles into

functional (muscular) collateral arteries concomitant with acquisition of elastic and vasomotor properties.[11]

Current studies describes the process of angiogenesis and arteriogenesis as newly differentiated vessels are due to endothelial cells migration, differentiation and multiplication to form complex, mature vascular network . [12]

Further study shows that vasculogenesis, an initial step in blood vessel formation in the fetus does not play a significant role in adult collateralization.

Based on the histological structure, the coronary collaterals has been classified into two categories:

- Capillary size collaterals without smooth muscle cells are predominantly seen in subendocardium
- Large muscular collaterals located in epicardium develops from preexisting arterioles. (uptodate coronary collateral circulation)[13]

Site of origin of collateral arteries

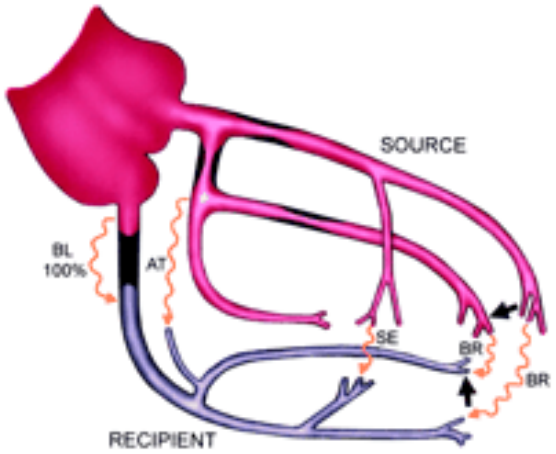
The collateral artery formed can orginate from different sites, as a

- terminal extension of two coronary arteries,
- branching out from the side of the two arteries,
- branching from the same stenosed artery,
- or within the same artery through vasa vasorum.

These type of collaterals occurs most commonly in

- ventricular septum
- ventricular apex
- ventricular free septum,
- anterolateral left ventricular free wall,
- cardiac crux,
- atrial surfaces.

- BL – bridging across lesion
- AT – atrial
- SE - septal
- BR – branch to branch in ventricular free wall



Determinants of Coronary Collateral Circulation

Factors determining the formation of collaterals

1. Recurrent and severe myocardial ischemia:

Most important determinant of development of Coronary collaterals is recurrent and severe myocardial ischemia [14]. Takeshita et al correlated intermittent myocardial ischemia with development of coronary collaterals and preserved even at rest and becomes functional when necessary.[15]

2. Duration of the ischaemia:

Herlitz et al compared patients with longer and short duration of angina pectoris before acute MI and showed that longer the duration, smaller the infarcts.[16]

3. Duration of ischaemia and collaterals

The coronary collaterals were increased in patients with longer duration of disease. Longer the duration of the disease, larger is the caliber of the vessel.

4. Timely enlargement of collaterals:

Enlargement of collaterals at the right time increases the period of golden hours and avoid death and MI in symptomatic patients. Sabia et al demonstrated that the myocardium in patients with acute MI may remain viable for prolonged period, in the presence of collaterals.[3]

5. Myocardial sensitivity:

Studies suggest that biochemical signals stimulate the development of coronary collaterals. These biochemical singals are the angiogenic growth factors, vascular growth endothelial growth factor mRNA which are released due to low oxygen levels. Buschmann & schaper suggested

that the growth of collaterals is not only dependent on ischemia, but also depend on other factor like shear stress.[17] Hypoxia induces angiogensis whereas shear stress induces arteriogenesis.

6. Pressure gradient and stress:

Increase shear stress mechanically mediates the process of arteriogenesis. Freedman SB et al stated that there exist a Pressure gradient difference between the portion of artery distal to occlusion and the collateral artery within 90s of occlusion, which provokes a inflammatory response. The inflammatory response causes damage to the internal elastic lamina and causes expansion of vessel. The maximal functional capacity of the collateral vessel is achieved at period of 6 months through structural remodeling.

Grading of collaterals:

Rentrop and colleagues were the first to describe the coronary angiographic method for collateral for spontaneously visible collaterals without artificial vascular occlusion(8). But later it was modified and the widely used method provides a score from 0–3 for recruitable collateral vessels upon occlusion of the ipsilateral artery [18]

MATERIALS AND METHODS

The present study of "clinical determinants of coronary collaterals" was carried out in the department of Cardiology, GGH, MMC, and Chennai.

400 Consecutive coronary angiogram done over a period of six months in the catheterization lab at GGH, MMC were observed. Among them 50 patients who had total or subtotal occlusion in coronary angiography were included in our study. Information regarding clinical presentation, risk factors, previous CAD, family history was meticulously recorded. This study was approved by the institute's ethical committee.

Permission from the hospital administration and consent from the patients was obtained for performing the study. Inclusion and exclusion criteria mentioned in the protocol were strictly followed and adhered.

I INCLUSION CRITERIA:

1. Age > 18 years

2. Known CAD patients who had undergone conventional coronary angiogram and had subtotal or total coronary artery occlusion on coronary angiography were included.

II EXCLUSION CRITERIA:

1. Age < 18 years

2. Patients with relative contraindications for coronary angiography which include active bleeding, coagulopathy, acute renal failure, chronic renal failure, active infection, uncontrolled hypertension, and severe anemia with Hb < 8gm, decompensated CCF were excluded.

III METHODS:

Patients with angiography showing subtotal or total coronary artery occlusion were assessed for

1. Age group:

50 Patient who formed the study group were classified into different age group

2. Risk factor:

A detailed history was elicited and documented for conventional coronary risk factor. The patients were evaluated for conventional risk factors like smoking, obesity, Diabetes mellitus, Systemic Hypertension, Family history of CAD, prior CAD, and sedentary lifestyle. The risk

factor was correlated with development of coronary collaterals on angiography as an isolated factor and as a multiple risk factor in patients with multiple coronary risk factors.

3. Clinical Presentation:

The patients with coronary occlusion were assessed for the mode of clinical presentation at the time of hospital admission prior to coronary angiography. They were grouped into 5 categories as ST elevation myocardial infarction(STEMI), non ST elevation myocardial infarction(NSTEMI), unstable angina (USA), chronic stable angina (CSA) and recurrent coronary artery disease(RECURRENT CAD).

4. Investigations:

Patients were investigated for hemoglobin, fasting and postprandial estimation of blood sugar, blood urea, serum creatinine. Standard 12 lead Electrocardiogram analyses were done in all patients. Echocardiographic evaluation was done for analysis of regional wall motion abnormalities and assessment of LV systolic function. A group of patients in our study group in whom ECG stress test was already done prior to coronary angiography were assessed for the correlation of stress test positivity and development of coronary collateral. They were separated into three

groups based on stage of stress test positivity by Bruce protocol for analysis of development of coronary collateral.

5. Coronary angiogram:

Detailed analysis of coronary angiograms of our study group was done. The parameters assessed were

i. number of total/ subtotal occluded vessels,

ii. types and

iii. segments of coronary artery involvement,

iv. presence or absence of coronary collateral arteries.

6. The parameters for coronary collateral arteries were assessed for its

i. number,

ii. Types of collaterals whether from branches of same coronary artery (homo collateral) or from branches of other coronary arteries (hetero collateral).

iii. Grading of collateral

On coronary angiogram, grading of coronary collateral was done by using Rentrop and cohen's collateral classification. Rentrop and cohen's grading of coronary collateral classification defines

Grade 0: No collateral arteries are present.

Grade 1: Barely detectable coronary flow. Contrast medium passes through collateral channels but fails to opacify the epicardial vessels at any time.

Grade 2: Partial collateral flow. Contrast material enters but fails to opacify the target epicardial vessel completely.

Grade 3: Complete perfusion. Contrast material enters and completely opacifies the target epicardial vessel.

The grade of collaterals was correlated with the following parameters

i. sex distribution
ii. age distribution
iii. risk factors
iv. clinical presentation
v. ECG stress test
vi. LV function

OBSERVATION

In this present study of clinical determinants of coronary collaterals 400 consecutives coronary angiograms performed at Madras Medical College were observed. Among the observed angiograms, patients with total or subtotal occlusion of coronary artery formed the study group. The following observation were made

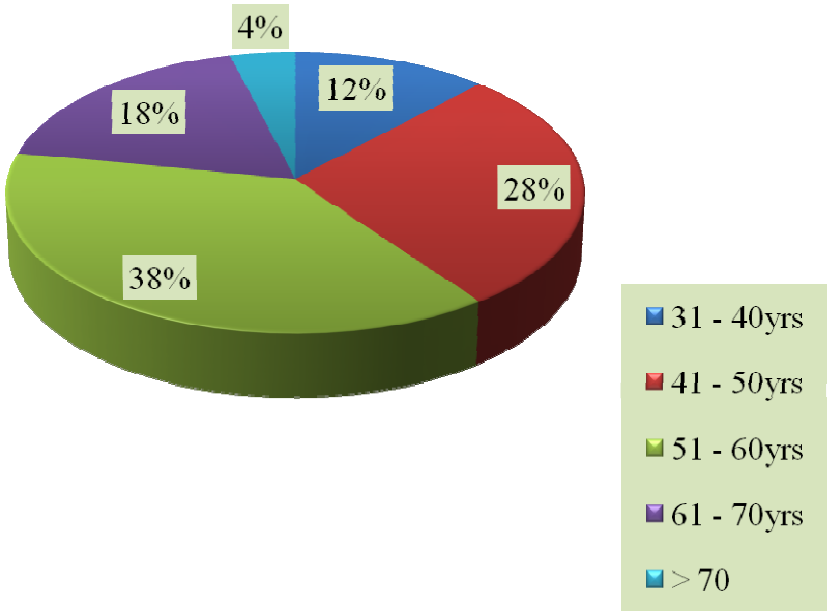
1. Distribution of cases:

A total of 50 patients with total or subtotal coronary artery occlusion formed the cases. The cases were categorized according to the age group as follows:

Table: I ***Distribution of cases***

S/No.	Age group (Yrs)	No of cases
1.	18 – 30	0
2.	31 – 40	6
3.	41 – 50	14
4.	51 – 60	19
5.	61 - 70	9
	>70	2
	Total no of cases	50

Chart I: Distribution of cases among age group in percentage



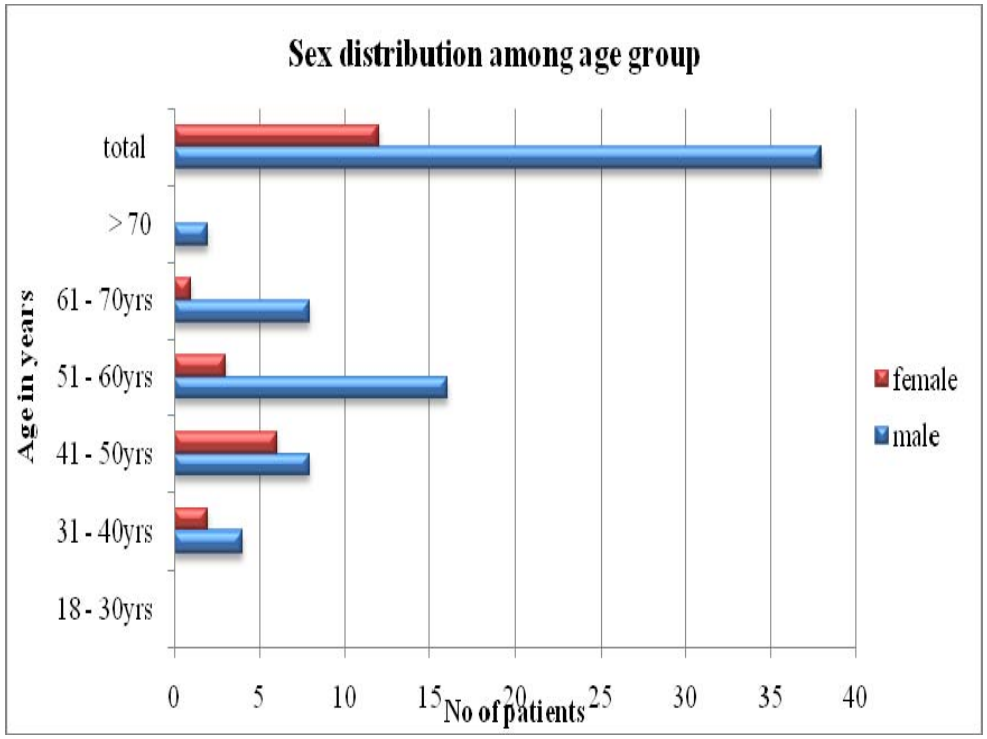
2. Sex distribution

Among the observed patients with coronary occlusion, the distribution of male and female patients were as follows:

Table: II *Sex distribution among cases*

S/No.	Age group (Yrs)	No of cases	
		Male	Female
1.	18 – 30	0	0
2.	31 – 40	4	2
3.	41 – 50	8	6
4.	51 – 60	16	3
5.	61 - 70	8	1
6.	>70	2	0
	Total no of cases	38	12

Chart II


Sex distribution among age group
total
> 70
61 - 70yrs
51 - 60yrs
41 - 50yrs
31 - 40yrs
18 - 30yrs
Age in years
0
5
10
15
20
25
30
35
40
No of patients
female
male

3. Distribution of clinical presentation:

The patients in the study group with occlusion of coronary angiogram were interviewed for condition at the time of admission, and were cross checked with case records present at Madras Medical College, the following clinical determinants were observed

Table III: ***Distribution of clinical presentation***

S/ No.	Clinical presentation	No of cases
1.	ST elevation MI	26
2.	NON ST elevation	6
3.	Unstable Angina	2
4.	Chronic Stable Angina	10
5.	Recurrent CAD	6
	Total	50

Chart III: Clinical presentation among cases

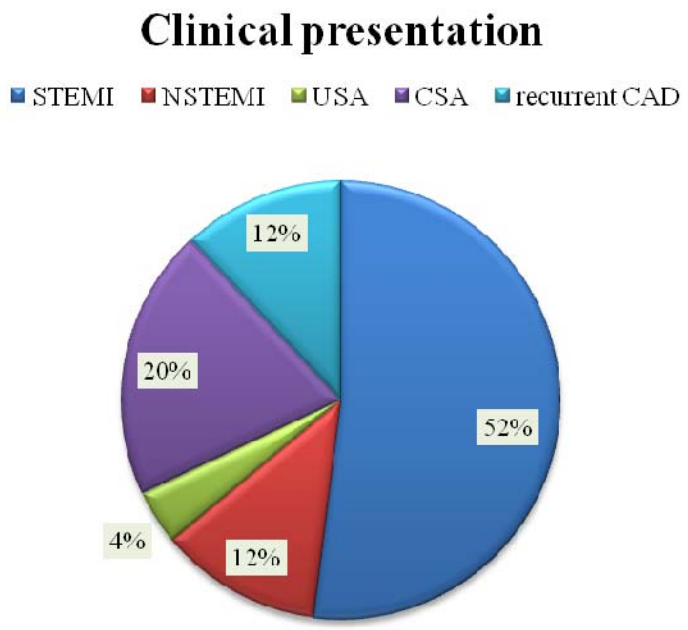
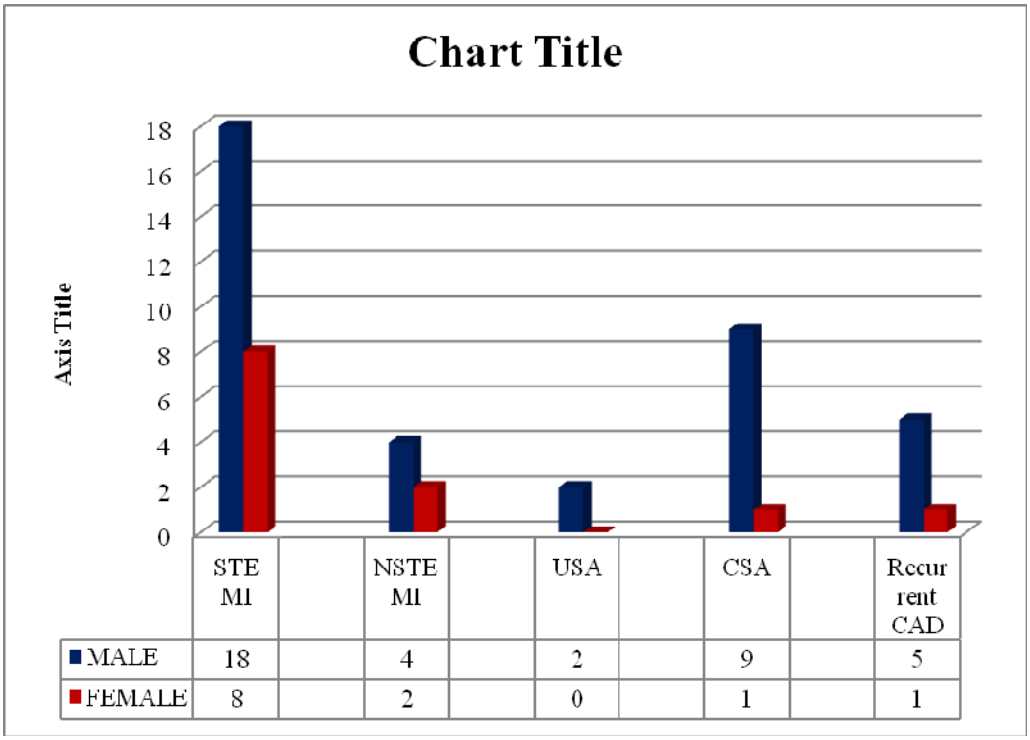


Table IV: C*linical presentation among Age groups*

	No. of Cases					
Age in years	**STEMI**	**NSTEMI**	**USA**	**CSA**	**Recurrent CAD**	**Total**
31 - 40yrs	**4**	**0**	**1**	**1**	**0**	**6**
41 - 50yrs	**7**	**3**	**1**	**2**	**1**	**14**
51 - 60yrs	**10**	**3**	**0**	**5**	**1**	**19**
61 - 70yrs	**5**	**0**	**0**	**1**	**3**	**9**
> 70	**0**	**0**	**0**	**1**	**1**	**2**
Total	**26**	**6**	**2**	**10**	**6**	**50**

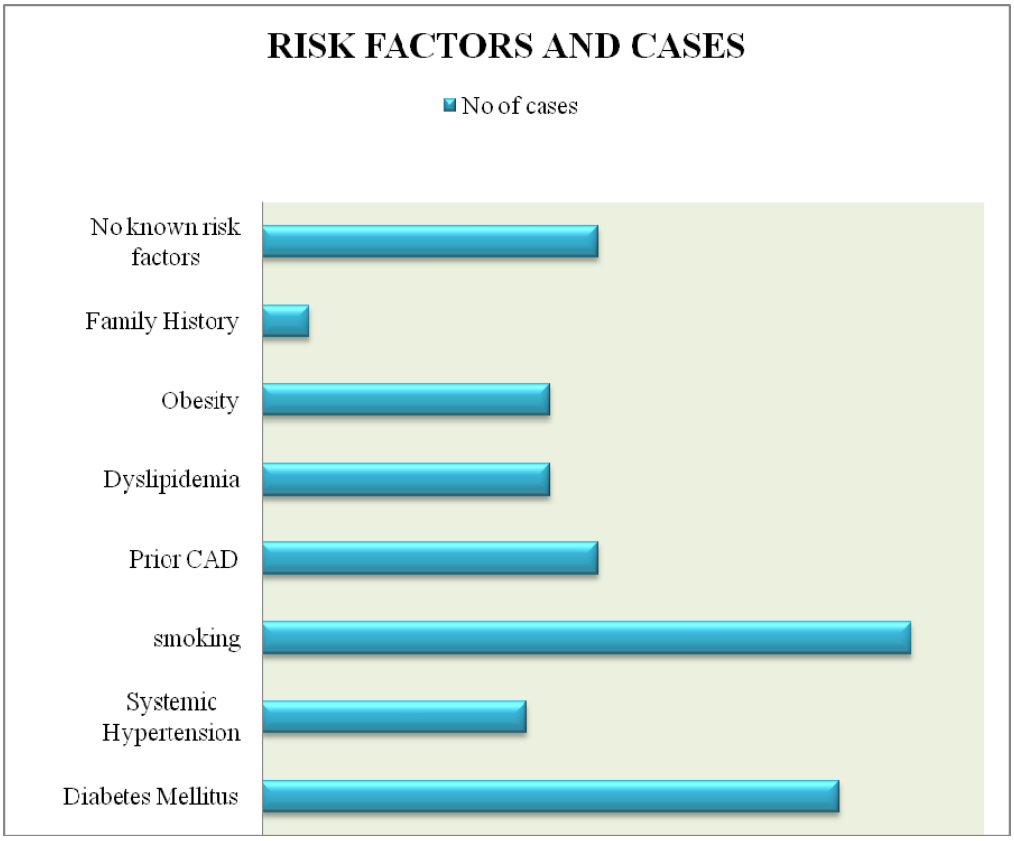
Chart IV: Clinical presentation among Male and Female



4. *Risk factors*:

The following conventional coronary risk factors in the patients were recorded by interview of patients and by case records

Chart V: Distribution of risk factors among cases



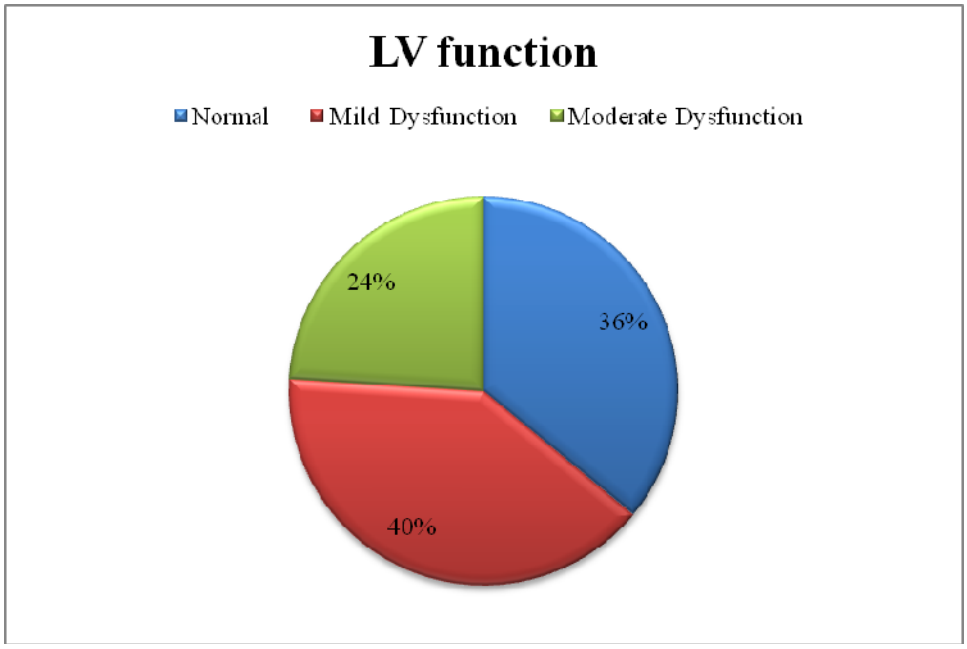
5. Left ventricle function:

All the patients in the study group were subjected to Echo to evaluate the left ventricle functional status. The patients were categorized into mild, moderate and severe LV Dysfunction based on the standard ECHO guidelines. The following information was recorded.

Table V: Distribution of cases according to LV function

Normal	18
Mild Dysfunction	**20**
Moderate Dysfunction	**12**
Severe Dysfunction	**No cases reported**

Chart VI: LV dysfunction in coronary artery occlusion



6. STRESS TEST:

Patients with who had undergone stress test prior to angiography and found positive were graded according to standard BRUCE protocol.

Table VI: No of cases in different stage of STRESS +ve test

TMT +VE	No Of Cases
STAGE I	**4**
STAGE II	**4**
STAGE III	**2**
Total	**10**

5. Description of coronary artery occlusion

The angiography of the study group patients were observed and documented**.** The coronary artery occluded among the 50 angiograms were classified into single artery occlusion and multiple artery occlusion, the details are as follows**:**

Table VII: Type of coronary artery occlusion

Coronary artery	**No of cases**		
	Male	**Female**	**Total**
Single artery			
LAD	**19**	**9**	**28**
LCX	**6**	**2**	**8**
RCA	**8**	**1**	**9**
Multiple artery			
LAD & RCA	**3**	**0**	**3**
LAD & LCX	**2**	**0**	**2**
		TOTAL	**50**

Chart VII: Distribution of single artery and multiple artery occlusion

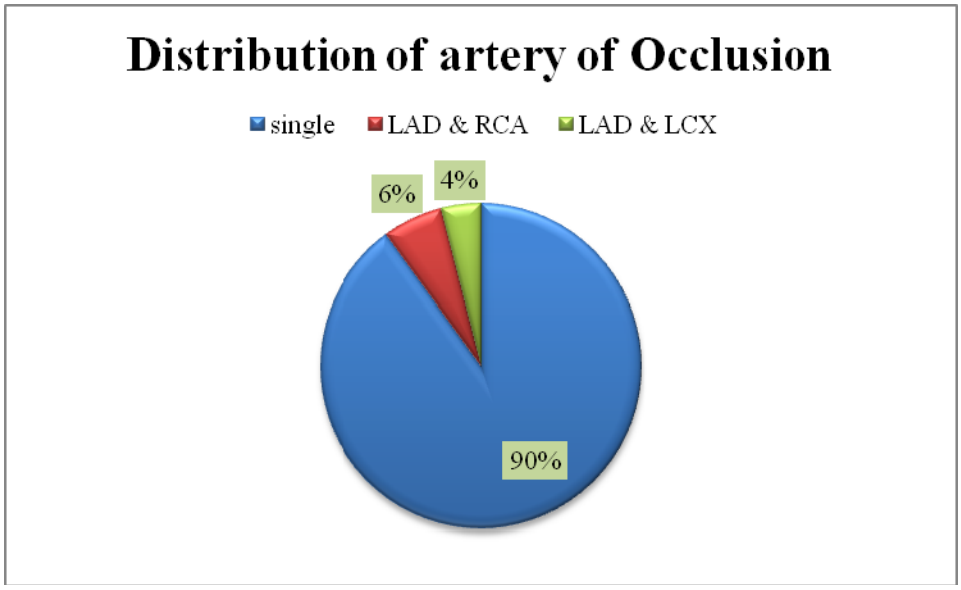
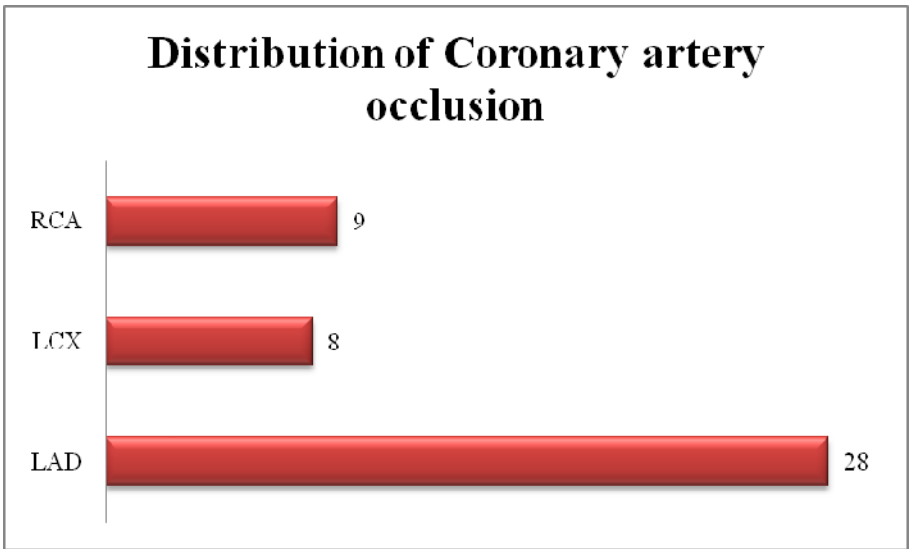


Chart VIII: Distribution of single coronary artery Occlusion



6. Site of occlusion of coronary artery

The site of occlusion of coronary artery was classified into proximal, middle and distal**.**

Table VIII: ***Type and segment of coronary artery occlusion***

	Type of Coronary artery		
Segment of Coronary artery	**LAD**	**LCX**	**RCA**
Proximal	**19**	**6**	**9**
Middle	**14**	**-**	**5**
Distal	**0**	**2**	**0**

7. *Coronary artery occlusion and grading of collateral*

The angiography with occlusion of coronary artery showed establishment of collaterals. The grading of collaterals was done according to standard protocol. For each independent and dual artery occluded, the artery from which the collaterals originated were studied and following observation were made

Chart IX: Grading of collaterals among cases

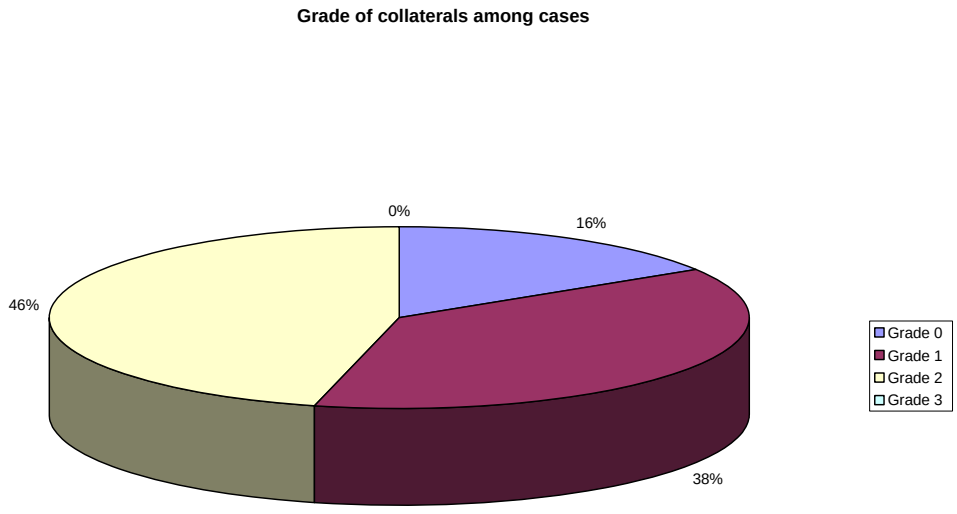


Table IX: LAD OCCLUSION AND COLLATERALS

Coronary artery	Homo	Hetero		Both	GRADING			
		LCX	RCA		0	1	2	3
Proximal	0	1	4	14	4	7	9	0
Middle	0	1	4	9	1	6	7	0
Distal	No cases reported							

Table X: LCX OCCLUSION AND COLLATERALS

Coronary artery	Homo	Hetero		Both	GRADING			
		LAD	RCA		0	1	2	3
Proximal	0	3	0	3	1	2	3	0
Distal	0	0	0	2	0	1	1	0

Table XI: RCA OCCLUSION AND COLLATERALS

Coronary artery	**Homo**	**Hetero**		**Both**	**GRADING**			
		LAD	**LCX**		**0**	**1**	**2**	**3**
Proximal	**0**	**4**	**0**	5	2	3	4	0
Middle	**1**	**3**	**0**	1	0	2	3	0
Distal	NO CASES REPORTED							

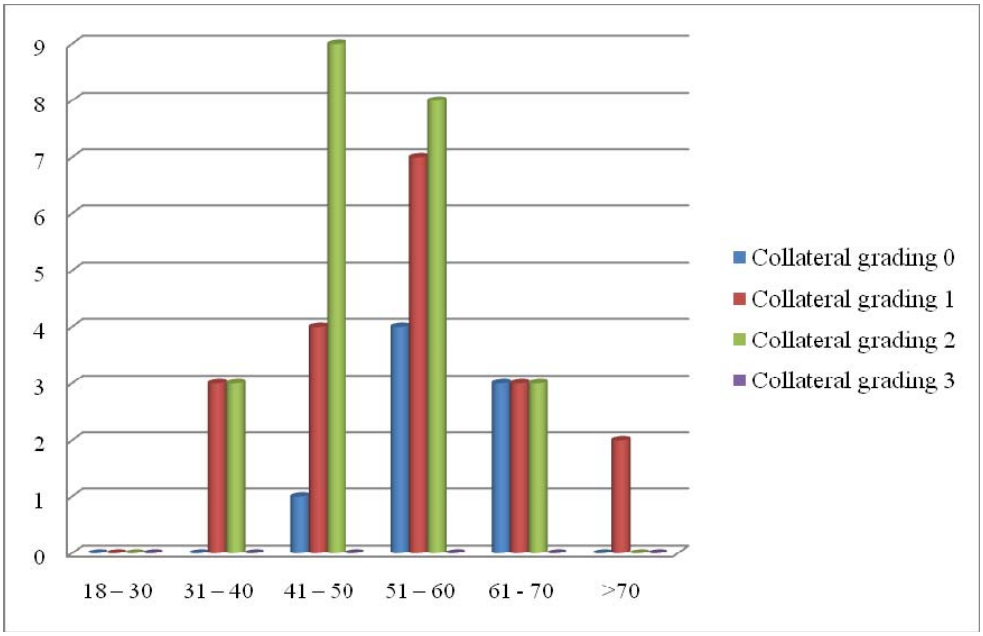
8. Collaterals and Age factors:

The collaterals established were graded and correlated with age groups

Table XII: AGE GROUP AND COLLATERALS

S/No.	Age group (Yrs)	Collateral grading			
		0	1	2	3
1.	18 – 30	0	0	0	0
2.	31 – 40	0	3	3	0
3.	41 – 50	1	4	9	0
4.	51 – 60	4	7	8	0
5.	61 - 70	3	3	3	0
6.	>70	0	2	0	0
	Total no of cases	8	19	23	0

Chart X: Age and collateral grading



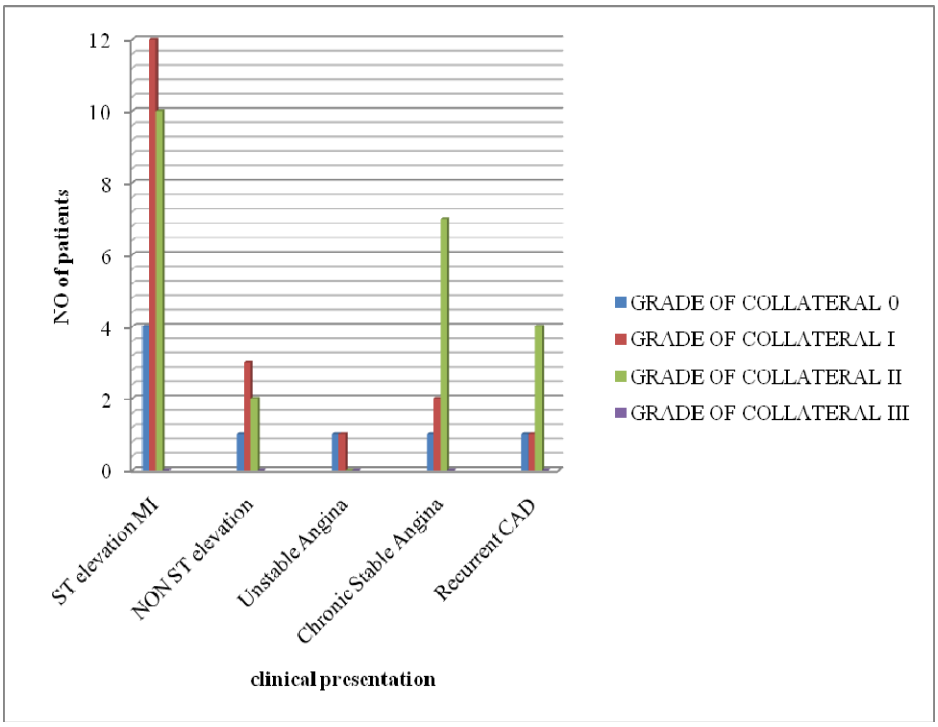
9. ***Collaterals and clinical presentation:***

The grading of collaterals formed were correlated with variety of clinical presentation the patients presented with at the time of admission

Table XIII: Clinical presentation and collaterals

S/ No.	Clinical presentation	GRADE OF COLLATERAL				Total no of Cases
		0	I	II	III	
1.	**ST elevation MI**	**4**	**12**	**10**	**0**	**26**
2.	**NON ST elevation**	**1**	**3**	**2**	**0**	**6**
3.	**Unstable Angina**	**1**	**1**	**0**	**0**	**2**
4.	**Chronic Stable Angina**	**1**	**2**	**7**	**0**	**10**
5.	**Recurrent CAD**	**1**	**1**	**4**	**0**	**6**
	Total	**8**	**19**	**23**	0	50

Chart XI: clinical presentation and grade of collaterals

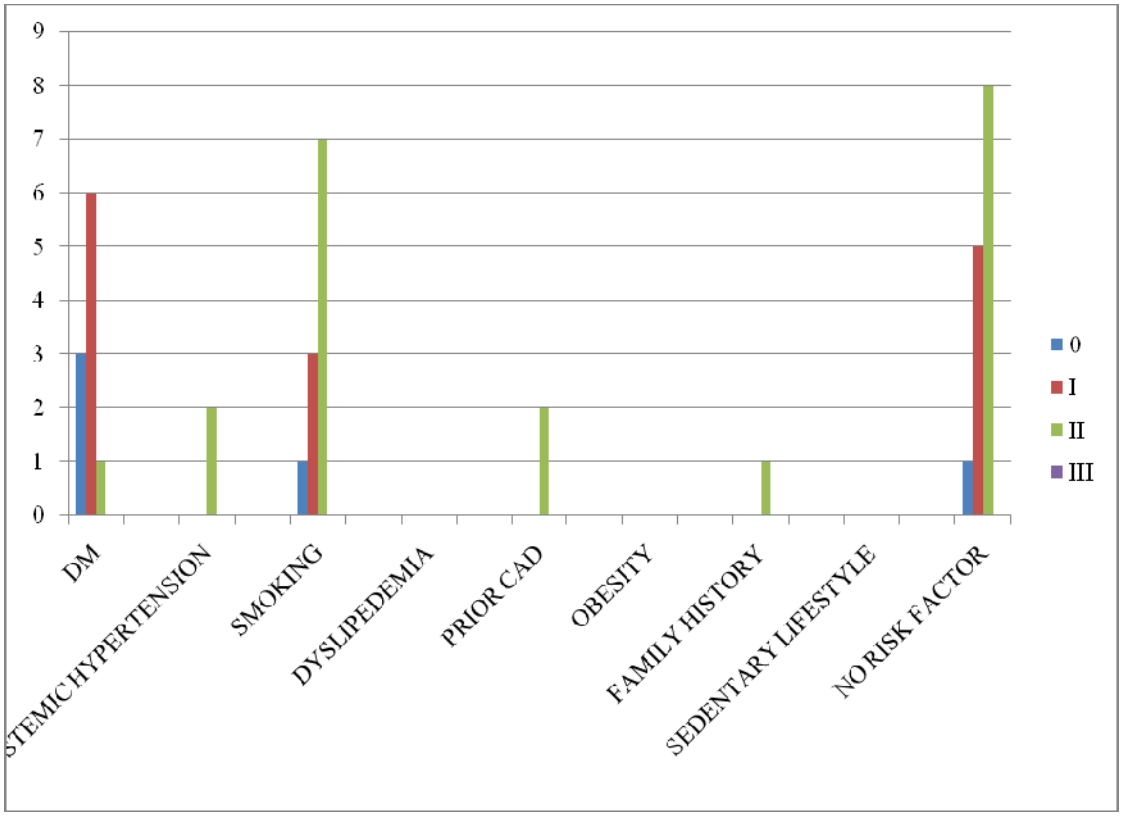


9. Risk factors and collaterals: The collaterals established were raded and correlated with variety of risk factor of the patients presented with at the time of admission

Table XIV: Risk factors and grade of collaters

S/ No.	RISK FACTORS	GRADING OF COLLATERAL				Total no of cases
		Isolated risk factor				
		0	I	II	III	
1	DM	3	6	1	0	10
2	SYSTEMIC HYPERTENSION	0	0	2	0	2
3	SMOKING	1	3	7	0	11
4	DYSLIPEDEMIA	0	0	0	0	0
5	PRIOR CAD	0	0	2	0	2
6	OBESITY					0
7	FAMILY HISTORY	0	0	1	0	1
8	SEDENTARY LIFESTYLE					0
9	NO RISK FACTOR	1	5	8	0	14
1	Multiple risk factors					
		3	5	2		10
		8	19	23	0	50

Chart XII: Isolated risk factors and grade of collaterals



11. Collaterals and sex distribution

The variations in collaterals established were analysed among different age group

Table XV: SEX DISTRIBUTION AND COLLATERALS

		0	1	2	3
1.	**Male**	**6**	**14**	**18**	**0**
2.	**Female**	**2**	**5**	**5**	**0**

12. LV function:

Table XVI: LV function and collaterals

S/No.	COLLATERAL GRADING				Total No of Cases
LV FUNCTION	**0**	**I**	**II**	**III**	
Normal	**3**	**7**	**8**	**0**	**18**
Mild LV dysfunction	**3**	**7**	**10**	**0**	**20**
Moderate LV dysfunction	**2**	**5**	**5**	**0**	**12**
Severe LV dysfunction	**No cases reported**				
Total	**8**	**19**	**23**	**0**	**50**

Patients who had undergone stress test were observed for collaterals.

Table XVII: Stress test and collaterals

TMT +VE	**No Of Cases**	**GRADE OF COLLATERAL**			
		0	I	II	III
STAGE I	**4**	**1**	**1**	**2**	**0**
STAGE II	**4**	**0**	**1**	**3**	**0**
STAGE III	**2**	**0**	**0**	**2**	**0**
Total	**10**	**1**	**2**	**7**	**0**

RESULTS

In our study a total of 50 patients with angiography showing total/subtotal occlusion formed the study group. Most of the patients presented to the hospital were in the age group of 51- 60yrs, followed by 41-50yrs (Table I). Among the 50 patients studied, 38 were males and 12 were females (Table II). Male predominantly (66%) presented in the age group >50 yrs whereas female presented early in life in the age group of 41- 50 years (chart I), but in all age group male were more in number than females (chart II).

The patients presented to the hospital in different modes of clinical presentation. Acute coronary syndrome accounted for 68% of the patients (chart III). ACS occurred most commonly in the age group of 40-60yrs (Table IV). Most common clinical presentation was STEMI (52%), followed by chronic stable angina (20%), NSTEMI (12%), recurrent CAD (12%) and unstable angina (4%) (chart III). Out of 26 patients with STEMI, 17 patients were in the age group of 40-60yrs. 70% of the Chronic stable angina occurred above the age group of 50yrs. Recurrent CAD predominantly occurred in the age group of 60-70yrs (Table IV). STEMI was a common clinical presentation both in males and females

(Chart IV). Unstable angina, CSA, recurrent CAD was more common in males than in females of the same age group.

There were many associated risk factors observed among the patients like smoking, diabetes mellitus, systemic hypertension, prior CAD, dyslipidemia, obesity, and family history. There were 14 patients (28%) without any known risk factors. 9 patients had two or more risk factors. Most common risk factor observed was smoking, followed by diabetes mellitus. Out of 38 males, 27 were smokers, with or without other associated risk factors (Chart V).

On ECHO, the status of Left ventricular function was analyzed, 36% of the patients had normal LV function, 40% had mild LV dysfunction, and 24% had moderate LV dysfunction (chart VI). Severe LV dysfunction was not noted in our study (Table V).

Among the 10 patients who had undergone STRESS TEST, 4 were in stage I, 2 patients were in stage II and 2 patients were in stage III (Table VI), according to the standard Bruce protocol.

Angiography of the 50 patients under study revealed single coronary or occlusion of both coronary arteries. 90% of the patients had single coronary artery occlusion, whereas occlusion two coronary arteries

were found only in 10% of the patient (Chart VII). LAD was the most common artery occluded both occurring in isolation or in association with occlusion of other artery (Chart VIII). The other arteries occluded were RCA AND LCX. In all the arteries occluded, proximal part of the artery was the commonly involved segment (Table VI).

Among the 50 patients under study, grade II collaterals was found in 46% of the patient, grade I collaterals was noted in 38% of the patients, no collaterals were noticed in 16% of the patients (Chart IX). Grade III collaterals were not noticed in any of our patients.

In both single artery occlusion and occlusion of two coronary artery, the collaterals were from both homo and hetero type of collateral (Table VII). LAD artery was the major contributor as collateral in both LCX and RCA type of occlusion. In LAD artery occlusion, RCA was the major contributor as collateral artery (Table VII, VIII, IX).

Grade 0 collateral was commonly observed in 50-70yrs of age. All patients above 70yrs of age had Grade I collateral. Both male and female patients had equal distribution of Grade of collateral (Table X).

Grade 0 collateral was found in 20% of patient with STEMI. Majority of the patients had Grade II collaterals. Grade II collaterals were

observed in 70% of patients with CSA and 66% of patients with recurrent CAD (Table XI).

Among 27 patients who were chronic smoker, 18 had grade II collateral, Grade 0 collateral were present in 5 out of 24 patients with DM mellitus. 50% of the patients with prior CAD had grade II collateral. Both the patients with positive family history of CAD had grade II collaterals. In patients with multiple risk factors, common collateral grade was Grade 0 and Grade1. Among 14 patients with no risk factors, 8 were found to have Grade 2 collateral (Table XII). 6 males and 2 females had Grade 0 collaterals (Table XIII)

There was no significant variation in collateral grades among normal and abnormal LV function (XIV).

Grade 0 collateral was noticed in 1patient among the 4 patients who had stage 1 positive stress test. Among the 6 patients with stage 2 & stage 3 positive stress test 5 patients had Grade 2 collaterals (XV).

DISCUSSION

CAD forms one of commonly occurring disease with multiple modes of presentation and risk factors, when detected early can reduce the mortality and morbidity. Coronary Collaterals which form an alternate source of blood supply forms vital as the presence of collaterals can delay the golden period and decrease the morbidity and increase the viable cardiac tissue.

Our study of clinical determinants of coronary collaterals was undertaken to establish the relevance of different variables like clinical presentation, Left ventricular systolic function, ECG stress test,conventional risk factors, age and sex with coronary collateral development.

In our study group of 50 patients with angiography showing total or subtotal occlusion of coronary artery, 76% were males. Females presented early around 40-50yrs of age, whereas males presented at a later age of 50yrs and above.

The modes of clinical presentation with the patients presented to hospital varied considerably. Two third of the patient presented with acute coronary syndrome. The commonest mode of presentation was

STEMI. STEMI presented commonly in the age group of 40-60yrs of age. Other modes of presentation in order of decreasing frequency in our study were chronic stable angina, NSTEMI, recurrent CAD and unstable angina. Chronic stable angina and recurrent CAD commonly presented between the age group of 50-60yrs and 60-70yrs of age respectively.

The most common associated risk factor in our study was smoking. Among male patients, 71% of them were smokers. 48% of the patients had Diabetes mellitus. Dyslipidemia was present only in 24% of our patients. 18% of the patients had multiple risk factors. History of prior coronary artery disease was present in 28% of patients. Systemic hypertension was seen in 22% of cases and a positive family history was present in 4% of cases. Interestingly 28% of the patients had none of the conventional coronary risk factors.

Piek JJ et al., observed angiographic study of single artery occlusion, where 69% of patients had LAD artery occlusion and 25% had right coronary artery occlusion[19]. In our study, 90% of the patients had single artery occlusion, among the single artery occlusion, 62% of the patients had LAD occlusion and 20% of the patients had RCA involvement which is in concordance with Piek JJ et al. But our study group had 18% of patient with LCX involvement which is double the

incidence of study of Piek JJ et al where only 6% of the patient had left circumflex artery involvement. In Robert WC etal study revealed proximal segment of the LAD and LCX are commonly involved segments [20]. In our study also proximal segment of the left coronary system was most commonly involved were commonly affected . Cosby RS etal ., study the proximal and mid portion of RCA was commonly involved with least distal occlusion .[21] In our study also proximal and mid portion of RCA are the commonly involved and none of patients had distal RCA occlusion.

Grading was done by Rentrop & cohen grading. In our study grade 2 collateral was the commonest grade seen in 46% of patients . Grade 2 collateral was more common in chronic CAD, recurrent CAD patients. No collateral was seen in 16 % of patients with two third of them occurred in diabetes mellitus patients.

Cosby RS etal. LAD occlusion had collaterals in majority of the cases and collaterals originated from multiple collateral channels. The primary source of inter coronary flow was from conus artery followed by posterior descending artery (PDA) and posterolateral branches(PLB). [21] Collaterals from Septal and diagonals are less common.

In our study group with LAD occlusion , collateral were seen in 85% of patients.Collaterals were separated as homocollateral (from either diagonal or septal branches) and heterocollateral from LCX(either from obtuse marginal or distal LCX) or from RCA(conus branch, PDA,PLB). The most common mode of collateral supply to LAD distal to occlusion is supplied by both homo collateral and heterocollateral together in 66% in proximal LAD and 65% in distal LAD occlusion. Only Heterocollaterals supplied the distal portion in remainder of the patients. none of the patients received only homocollaterals. RCA was the most common artery supplying collateral to LAD. All our study findings were in concordance with the corby RS etal.study.

In LCX occlusion , collaterals were formed in 7 out of 8 cases with grade 2 collateral in 50% of them. Both homo collateral and heterocollateral together in 50% in proximal LCX and 100% of distal LCX occlusion . Only Heterocollaterals supplied the distal portion in remainder of the patients. LAD was the major source collateral supply to LCX .

In RCA occlusion, collaterals were found in 13 out of 14 patients. Isolated Hetero-collaterals were commonly found in RCA occlusion than LAD and LCX. Only one patient out of 50 patients had Homo-collateral,

which was present in RCA occlusion. Most of the collaterals for RCA was from LAD.

Age and collateral:

In our study, Grade 0 (No collaterals) was observed in 25% of the patients belonging to age group of 50 – 70yrs, and 36% had grade I collaterals and rest with grade II collaterals. two patients were in the age group above 70yrs and possessed grade I collaterals. Kurotobin et al., observed that the prevalence of collaterals decreases with advancing age was in concurrence with our study.

Clinical presentation and collateral:

In patients with STEMI & NSTEMI presentation, 84%had presence of collaterals. Grade 1 collaterals were the commonest type of collaterals ,whereas in the total study group grade 2 collaterals were the common collateral.

In patients with unstable angina, 50% of the patients had Grade I and 50% had Grade 0 collateral.

Meir P et al. in a 10yr follow up study found that long term cardiac mortality is decreased with patients with chronic stable angina due to

increased functional collaterals.[23] Mills JD and others found in animal studies that collaterals were well developed in chronic canine model. Finding in our study patients with chronic stable angina was in concurrence with above study. collaterals were found in 90% of the patients. 70% had grade II collaterals. Recurrent CAD patients, 84% of the patients had presence of collaterals. 66% of patients had grade II collaterals, whereas total study group, 46% had grade II collaterals, indicating that previous angina could trigger the formation of collaterals, these findings are in concordance with Fujita M et al., found that Collateral channels were present in two of 19 patients without angina before infarction and nine of the 18 patients with angina before infarction.[24]

Patients with acute coronary syndrome had predominantly grade I collaterals, whereas recurrent CAD and chronic stable angina had predominantly grade II collaterals

Risk factors and collaterals

In 90% of the patients with smokers had presence of collaterals, 60% had Grade II collaterals.

Abaci et al, 1999, observed that there were more number of diseased vessels in patients with diabetes mellitus and less number of collaterals.[25]

In our study, patients with isolated risk factor as DM had 30% of grade 0 collaterals and 60% of the patients had Grade I collaterals. 10% of the patients with DM had grade II collaterals in comparison with 46% of the total study population with grade II collaterals suggesting a poor development of collaterals in diabetes patient in concordance with the above study by Abaci et al.

Zenon S et al., studied that patient with systemic hypertension and CAD had increased coronary collateral circulation in correlation with LV wall thickness. In our study, two patients had systemic hypertension. Both presented Grade II collaterals coincides with study of Zenon et al.

Only one patient had presented with isolated family history of coronary heart disease. The patient presented as with STEMI possessed grade II collaterals.

28% of our patients presented without any conventional risk factors. 92% of the patients had coronary collaterals. 57% had grade II collaterals.

20% of our total study group had multiple risk factors. 30% of the patients had grade 0 collaterals and 50% grade I collaterals. 20% had grade II collaterals suggesting poorly developed collaterals.

There was no significant difference in grade of collaterals between male and female.

LV function and collaterals:

Hansen JF (1989) and Nacolau JC et al.,(1997) analysed the relationship between collateral flow and LV systolic function and found improved LV function in patients with grade III collaterals. [26]we could not correlate between LV function as our patient did not present with Grade III collaterals. Patients with Grade II and I collaterals did not have much difference in LV function.

ECG stress test and collaterals

Akutsu Y et al., in their described that exercise reduce the blood flow through collateral dependent area. In our study patients with stage I positive stress test had grade 0, grade I, grade II collaterals, whereas patients with stage II and III positive stress test had grade II collaterals, suggesting that patients with grade II collaterals can have more tolerance to exercise.

SUMMARY

- This study was carried out at department of cardiology, Madras Medical College, Chennai.
- Fifty patients with total/subtotal coronary artery occlusion on coronary angiography formed the study group.
- Various variables like clinical presentation, Left ventricular systolic function, ECG stress test, conventional risk factors, age and sex were studied and correlated with coronary collateral development.
- The collaterals were analyzed on coronary angiography.
- The grading of collaterals were correlated with variables
- The study group had more number of males than females
- The predominate age of presentation were in the age group of 40-60yrs of age
- The modes of clinical presentation was STEMI, NSTEMI, Unstable angina, Chronic stable angina, and recurrent CAD.

- STEMI was the most common clinical presentation.
- Associated risk factors like smoking, DM, systemic Hypertension, dyslipidemia, lifestyle, obesity, prior CAD, family history of CAD was evaluated. The risk factors were categorized into isolated risk factors and multiple risk factors. Most common associated risk factor was smoking.
- On angiographic study, LAD was the most common artery occluded, followed by LCX and RCA.
- Proximal segment of the coronary artery were the commonly involved segment.
- Collaterals were graded according to Rentrop and cohen classification on angiography.
- Grade II collateral was the commonest collateral observed
- Both homo and hetero collateral together were the mode of collateral supply in LAD occlusion. Branches from RCA was most the most common collateral in LAD occlusion.

- Both homo and hetero collateral together were the mode of collateral supply in LCX occlusion, Branches from LAD was most the most common collateral in LCX occlusion

- In RCA occlusion, hetero collaterals were the commonest mode of supply. Isolated homo collateral was seen in RCA occlusion. Branches from LAD was most the most common collateral in RCA occlusion

- Grade II collateral was commonly seen in age groups below 70yrs of age, and Grade I above 70yrs of age.

- In acute coronary syndrome, Grade I collaterals were the commonest collateral.

- Most of the STEMI AND NSTEMI patients had coronary collaterals and were predominantly grade I collaterals.

- In chronic stable angina, collaterals were found in majority of patients, mostly Grade II collaterals.

- In Recurrent CAD, collaterals were found in majority of patients, mostly Grade II collaterals

- In patients who were smokers, most of the patient had collaterals. Grade II collaterals were commonly observed.

- Majority of the patients with isolated risk factor of DM had Grade 0 and Grade I collaterals.

- In patients with systemic hypertension, grade II collaterals were commonly seen.

- Isolated family history, grade II collaterals was seen

- Majority of the patients with multiple risk factor of DM had Grade 0 and Grade I collaterals

- There was no correlation between LV function and grade of collaterals.

  No patients had Grade III collaterals

- In ECG Stress test, stage II & III positive patients had predominantly grade II collaterals.

CONCLUSION

- STEMI was the most common clinical presentation
- LAD is the most common artery occluded.
- Both homo and hetero collateral together were the commonest mode of collateral supply.
- Collateral to LAD most commonly arise from RCA
- Collateral to RCA and LCX most commonly arise from LAD
- The predominance of Grade II collaterals were observed in patients with CSA, recurrent CAD, smokers, systemic hypertension and patient with no conventional risk factors.
- Poorly formed collaterals (Grade 0 & Grade I) were observed STEMI, NSTEMI, unstable angina, DM, multiple risk factors, age > 50yrs.

BIBLIOGRAPHY

1. Popma JJ, Bittl J. Coronary angiography and intravascular ultrasonography. In: Braunwald E, Zipes DP, Libby P, eds. Heart Disease: A Textbook of Cardiovascular Medicine. Philadelphia: W.B. Saunders Company; 2001: 387–418.

2. Fukai M, Ii M, Nakakoji T, et al. Angiographically demonstrated coronary collaterals predict residual viable myocardium in patients with chronic myocardial infarction: a regional metabolic study. J Cardiol. 2000; 35: 103–111

3. Sabia PJ, Powers ER, Ragosta M, et al. An association between collateral blood flow and myocardial viability in patients with recent myocardial infarction. N Engl J Med. 1992; 327: 1825–1831

4. Fulton WFM. The time factor in the enlargement of anastomoses in coronary artery disease. Scot Med J. 1964; 9: 18–23

5. Conway EM, Collen D, Carmeliet P. Molecular mechanisms of blood vessel growth. Cardiovasc Res. 2001; 49: 507–521

6. Thomas A.gaziano Robert O..bonow, douglas L.mann, eds. Heart Disease: A Textbook of Cardiovascular Medicine. Philadelphia: W.B. Saunders Company; 2011:1-2

7. Joseph k.malouf, fuster , walsh HURST‘THE HEART 13 th edition pages 84-86.

8. BolingerM etal. Is the development of myocardial tolerance to repeat ischemia in humans due to preconditioning or to collateral recruitement?aj am coll cardio1999;33;1027-35.

9. Baroldi G, Mantero O, Scomazzoni G. The collaterals of the coronary arteries in normal and pathologic hearts. Circ Res. 1956; 4: 223–229.

10. Schaper W, Gorge G, Winkler B, et al. The collateral circulation of the heart. Prog Cardiovasc Dis. 1988; 31: 57–77

11. Carmeliet P. Mechanisms of angiogenesis and arteriogenesis. Nat Med. 2000; 6: 389–395.

12. Conway EM, Collen D, Carmeliet P. Molecular mechanisms of blood vessel growth. Cardiovasc Res. 2001; 49: 507–52

13. wernerGS, ferrariM , etal. Collateral in chronic total occlusions, circulation 2001;104;2074

14. Sasayama S, Fujita M. Recent insights into coronary collateral circulation. Circulation. 1992; 85: 1197–1204.

15. Takeshita A, Koiwaya Y, Nakamura M, et al. Immediate appearance of coronary collaterals during ergonovine-induced arterial spasm. Chest. 1982; 82: 319–322.

16. Herlitz J, Karlson BW, Richter A, et al. Occurrence of angina pectoris prior to acute myocardial infarction and its relation to prognosis. Eur Heart J. 1993; 14: 484–491

17. Schaper W, Buschmann I. Arteriogenesis, the good and bad of it. Cardiovasc Res. 1999; 43: 835–837

18. Rentrop KP,cohen M etal changes in collateral channel filling after coronary artery occlusion by an angiop[lasty balloon in human subjects j am coll cardio 1885;5;587-592.

19. Piek JJ, van Liebergen RA, Koch KT, Peters RJ, David GK Clinical, angiographic and hemodynamic predictors of recruitable collateral flow assessed during balloon angioplasty coronary occlusion

20. Werner GS, Ferrari M, Betge S, Gastmann O, Richartz BM, Figulla HR TI Collateral function in chronic total coronary occlusions is related to regional myocardial function and duration of occlusion

21. Cosby RS, Giddings JA etal. Clinic arteriographic correlation in angina with and without myocardial infarction

22. Kurotobi T, Sato H, Kinjo K, Nakatani D, Mizuno H, Shimizu M, Imai K, Hirayama A, Kodama K, Hori M, OACIS Group Reduced collateral circulation to the infarct-related artery in elderly patients with acute myocardial infarction.

23. Meier B, Seiler C SO Beneficial effect of recruitable collaterals: a 10-year follow-up study in patients with stable coronary artery disease undergoing quantitative collateral measurements Circulation. 2007;116(9):975

24. Fujita M, Sasayama S, Ohno A, Nakajima H, Asanoi H Importance of angina for development of collateral circulation

25. Abaci A, Oğuzhan A, Kahraman S, Eryol NK, Unal S, ArinçH, Ergin A Effect of diabetes mellitus on formation of coronary collateral vessels

26. Nicolau JC, Pinto MA, Nogueira PR, Lorga AM, Jacob JL, Garzon SA The role of antegrade and collateral flow in relation to left ventricular function post-thrombolysis.

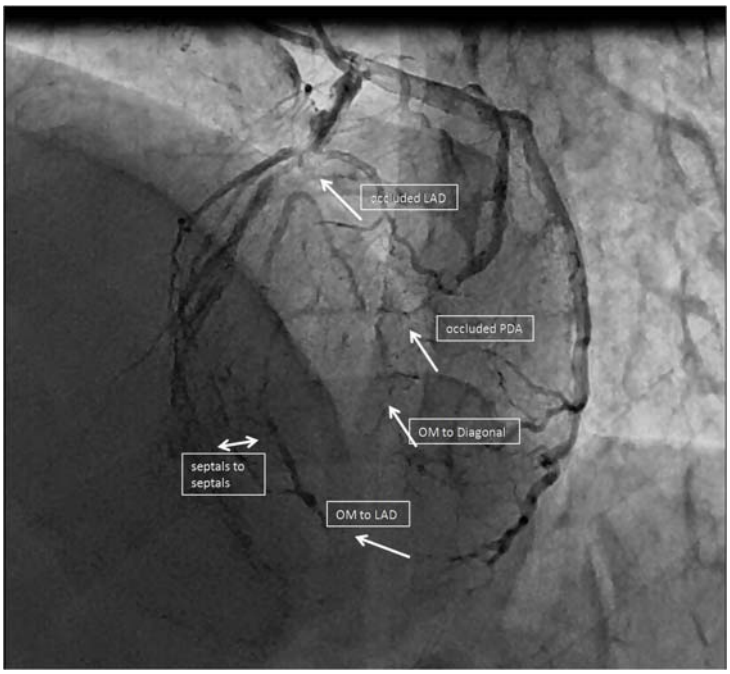
MASTER CHART

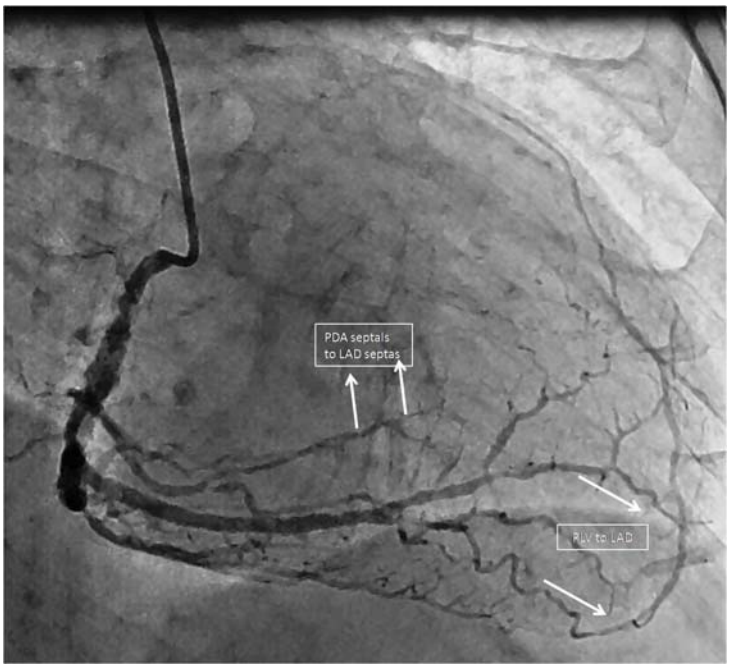
S. N.	Name	Risk factor	Age	Sex	Clinical diagnosis	Angio NO.	Coronary artery			COLLATERALS			Ejection fraction (EF)
							LAD	LCX	RCA	Type	Origin	Grade	
1.	Suryanarayanan	Dm/smo/ Prior	58	M	STEMI	1706		+		HETERO	LAD &RCA	2	36
2.	Haridas	nil	53	M	USA/	1839		+		nil		0	N
3.	Swadesi	Dm/Smo/Dys	40	M	STEMI	1665	+			Hetero	RCA	2	48
4.	Gopinath	DM/SHT/ Prior CAD	57	M	STEMI	1573	+			Hetero	RCA	1	42
5.	Pakkirisamy	Nil	60	M	STEMI	1515	+			Both homo and hetero	LCX/LAD	1	46
6.	Savithra	DM	38	F	STEMI	1751	+			Hetero	RCA	1	46
7.	Ganesan	DM	55	M	STEMI	1800			+	Both hetero/homo	LAD	2	48
8.	Suman	SMO	48	M	NSEMI	1764	+			Both homo and hetero	RCA/LAD	2	49
9.	chandrasekaran	nil	45	M	CSA/ tmt +	1812	+			Both homo and hetero	LCX/RCA	2	55
10.	Kala	SMO	38	F	STEMI	1857		+		heterocollateral	LAD	1	55

11.	Ravi	DM	45	M	STEMI	1703	+		+	nil		0	58
12.	Ruche	SM/HT	65	F	USA	1711	+			heterocollateral	RCA	2	38
13.	Kumar	DM	67	M	CSA	1765		+		Both hetero/homo	LAD/RCA	2	36
14.	Chitra	NIL	62	F	REC.CAD	1867	+			Both hetero/homo	RCA/LAD	1	46
15.	Saravanan	NIL	63	M	STEMI	1550			+	Both hetero/homo	LAD	2	48
16.	Ekambaram	SMO	57	M	STEMI	1729		+		Both hetero/homo	LAD	1	60
17.	Sreenivas	SMO	46	M	CSA	1831	+			nil		0	42
18.	Gunasekarasn	SMO/DM	48	M	REC.CAD	1871	+			heterocollateral	RCA	2	48
19.	Sundaram	SMO/OBE	58	M	STEMI	1864			+	Both hetero/homo	LAD	2	56
20.	Narayanan	SHT/DM	57	M	CSA	1543	+	+		nil		0	40
21.	Murugesan	SMO/DM	53	M	REC.CAD	1501			+	Both hetero/homo	LAD	1	48
22.	Latha	PRIOR CAD	69	F	NIL	1509	+			heterocollateral	RCA	1	50
23.	Hari	SMO	37	M	CSA	1753	+		+	Both hetero/homo	LAD/LCX	2	38
24.	Muniyammal	DM/SHT	38	F	STEMI	1821		+		Both hetero/homo	LAD	2	56

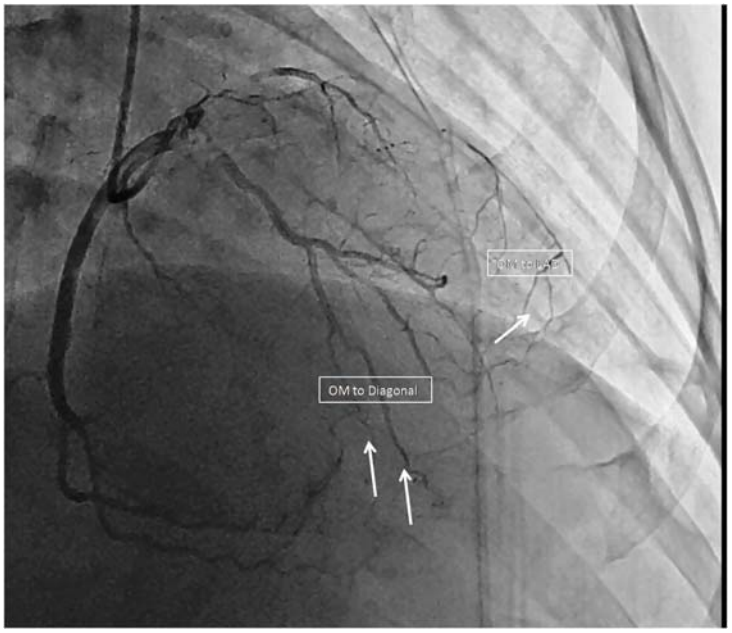
25.	Kamakchi	SHT/DM	46	F	NSTEMI	1859	+			nil		0	55
26.	Chandran	SMO	42	M	STEMI	1653			+	Both hetero/homo	LAD/LCX	1	42
27.	Ambikapathi	SMO/DM	57	M	USA	1622	+			Both hetero/homo	LCX	2	38
28.	Durai	SMO/DM	55	M	STEMI	1576	+		+	nil		0	52
29.	Rajkumar	SMO	67	M	CSA	1669		+		Both hetero/homo	LAD	1	50
30.	Visalakshi	DM	63	F	REC.CAD	1798	+			Both hetero/homo	LCX/RCA	2	56
31.	Dayadharan	OBE/SMO	55	M	STEMI	1799			+	homocollateral	Conus branch RCA	2	58
32.	Sreenivasan	FAM H/O	70	M	NSTEMI	1798	+			heterocollateral	LCX	1	46
33.	Murugaraj	SMO	75	M	STEMI	1551	+			heterocollateral	RCA	1	48
34.	Meenal	DM/SHT	42	F	CSA	1534		+		nil		0	58
35.	Nagaraj	SMO/DM	57	M	REC.CAD	1653			+	heterocollateral	LAD	2	55
36.	Laksmi	OBE/SMO	68	F	STEMI	1789	+			heterocollateral	LCX/RCA	1	42
37.	Balu	DM/SMO	63	M	STEMI	1834		+		Both hetero/homo	LAD/OM	1	46
38.	Chinraj	DM/FAMH/O/SHT	48	M	STEMI	1823	+			nil		0	40

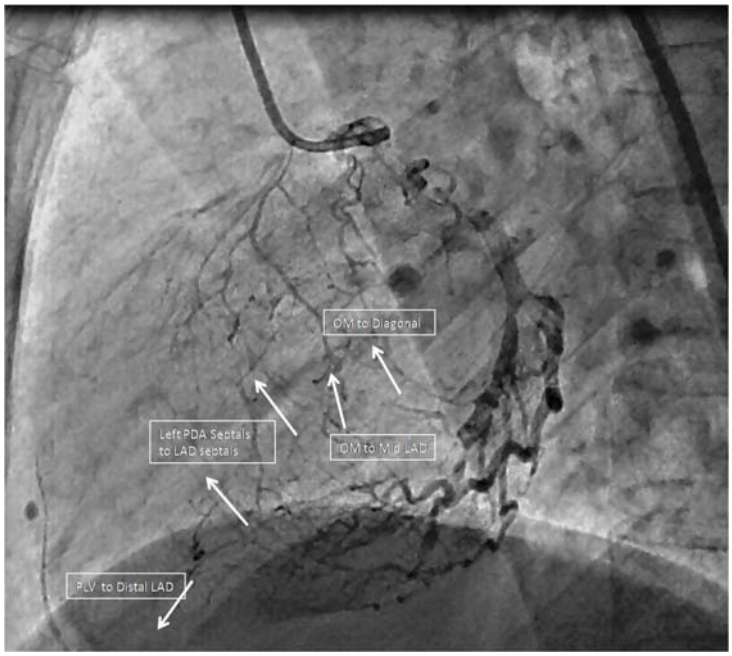
39.	Ragupathi	SMO/SHT	M	39	NSTEMI	1799			+	heterocollateral	1	56
40.	Laksman	DM/SMO	M	56	STEMI	1558	+			nil	0	48
41.	Beemn	SMO	M	48	CSA	1545	+			Both hetero/homo	2	50
42.	Kumuda	DM	F	68	STEMI	1826			+	Both hetero/homo	2	46
43.	Nagendrabau	PRIOR CAD/SMO	M	65	NSTEMI	1744	+			heterocollateral	1	42
44.	Veeraiya	SMO/PRIOR CAD	M	59	CSA	1894	+			Both hetero/homo	1	50
45.	Chinnasamy	SMO	M	51	STEMI	1508		+		Heterocollateral;	2	56
46.	Meenakshi	DM	F	49	REC.CAD	1503	+			heterocollateral	2	55
47.	Guru	DM/SMO	M	68	CSA	1590			+	nil	0	42
48.	Gopal	DM	M	52	STEMI	1578	+			heterocollateral	1	40
49.	Balu	SMO	M	47	NSTEMI	1540	+			Both hetero/homo	2	46
50.	Rajui	SMO/DM	M	64	REC.CAD	1644			+	Both hetero/homo	2	48





ANGIOGRAPHIC PICTURES





ANGIOGRAPHIC PICTURES

PROFORMA

NAME: AGE: yrs SEX: M / F

IP NO:

HT: WT: BMI:

ADDRESS:

ANGIO DATE & NO:

PRESENTATION:

1.STEMI

2. NSTEMI

3.USA

4. CSA

5.RECURRENT CAD

DURATION OF CAD:

RISK FACTORS :

1. IGT 2. DM 3. SHT

4. PRIOR CAD 5.SMOKING 6. DYSLIPEDEMIA

7. FAMILY H/O CAD 8.OBESITY

9. SEDENTARY LIFESTYLE

INVESTIGATIONS:

HB: BL.SUG: FS: PP[2HRS]:

HBA1C:

BL.UREA: Sr.CR:

TMT:

CORONARY ANGIOGRAM:

LMCA-

LAD-

LCX-

RCA-

NO.OF TOTALLY/SUBTOTALLY >95% OCCLUDED VESSELS:

ANGIOGRAPHIC DIAGNOSIS:

COLLATERALS:

NUMBER:

HOMOCOLLATERLS:

HETEROCOLLATERALS:

TYPESOF COLLATERALS:

GRADING OF COLLATERALS:

Informed consent form

Title of the study -
Name of the participant: _____

Name of the Principal/Co-Investigator: _____

Name of the Institution: _____

Name and address of the sponsor / agency(ies), if any: _____

I,_____(name of participant), have read the information in this form (or it has been read to me). I was
free to ask any questions and they have been answered. I am over 18 years of age and, exercising
my free power of choice, hereby give my consent to be included as a participant in ___ " (title of the study)

(1) I have read and understood this consent form and the information provided to me.

(2) I have had the consent document explained to me.

(3) I have been explained about the nature of the study.

(4) I have been explained about my rights and responsibilities by the investigator.

(5) I have informed the investigator of all the treatments I am taking or have taken in the past _____ months including any native (alternative) treatments.

(6) I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in the hospital.

(7) I hereby give permission to the investigators to release the information obtained from me as result of participation in this study to the sponsors, regulatory authorities, Government agencies, and ethics committee. I understand that they may inspect my original records.

(8) I understand that my identity will be kept confidential if my data are publicly presented.

(9) I have had my questions answered to my satisfaction.

(10) I consent voluntarily to participate as a participant in the research study.

I am aware, that if I have any questions during this study, I should contact the investigators. By signing this consent from, I attest that the information given in this document has been clearly explained to me and understood by me. I will be given a copy of this consent document.